STATUS OF THE CLAIMS

1-33. (canceled).

34. (currently amended) A method of treating type II diabetes mellitus~~a disease~~, comprising:

a) providing:

i) a subject, wherein said subject suffers from type II diabetes mellitus~~a disease~~, wherein said type II diabetes~~a disease~~ comprises cells having increased cellular energy, increased mTOR function, and increased phosphorylated-S6K~~a defective cellular energy status~~;

ii) an agent; wherein said agent is an mTOR inhibitor~~reduces cellular ATP levels~~; and

b) administering said agent to said subject; wherein said agent targets said cells, wherein said targeting reduces said increased mTOR function and reduces said increased phosphorylated-S6K~~having a defective cellular energy status~~.

35. (previously presented) The method of Claim 34, wherein said agent is rapamycin.

36. (previously presented) The method of Claim 34, wherein said agent is a hexokinase inhibitor.

37. (previously presented) The method of Claim 34, wherein said agent is 2-deoxy-glucose.

38. (previously presented) The method of Claim 34, wherein said agent is a PKC inhibitor.

39. (previously presented) The method of Claim 34, wherein said agent is Rottlerin.

40. (previously presented) The method of Claim 34, wherein said agent is 5-aminoimidazole-4-carboxyamide ribonucleotide.

41. (previously presented) The method of Claim 34, wherein said agent is mitochondrial uncoupler FCCP.

42. (currently amended) The method of Claim 34, wherein said increased mTOR function, and increased phosphorylated-S6K~~defective cellular energy status~~ is caused by a mutation in the lkb-1 gene.

43. (currently amended) The method of Claim 34, wherein said increased mTOR function, and increased phosphorylated-S6K~~defective cellular energy status~~ is caused by a defective element of the cellular energy pathway, wherein said defective element is AMPK.

44. (currently amended) The method of Claim 34, wherein said increased mTOR function, and increased phosphorylated-S6K~~defective cellular energy status~~ is caused by a defective element of the cellular energy pathway, wherein said defective element is TSC2.

45. (currently amended) The method of Claim 34, wherein said increased mTOR function, and increased phosphorylated-S6K~~defective cellular energy status~~ is caused by a defective element of the cellular energy pathway, wherein said defective element is mTOR.

46. (canceled).

47. (currently amended) The method of Claim 34, wherein said type II diabetes mellitus~~disease~~ comprises complications~~is complications~~ associated with type II diabetes mellitus.

48. (canceled).

49. (previously presented) The method of Claim 47, wherein said complications associated with type 2 diabetes mellitus comprise renal dysfunction.

50. (canceled).